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NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2		"Ask CAS" for self-help around the clock
NEWS 3	FEB 27	New STN AnaVist pricing effective March 1, 2006
NEWS 4	MAY 10	CA/CAPLUS enhanced with 1900-1906 U.S. patent records
NEWS 5	MAY 11	KOREAPAT updates resume
NEWS 6	MAY 19	Derwent World Patents Index to be reloaded and enhanced
NEWS 7	MAY 30	IPC 8 Rolled-up Core codes added to CA/CAPLUS and USPATFULL/USPAT2
NEWS 8	MAY 30	The F-Term thesaurus is now available in CA/CAPLUS
NEWS 9	JUN 02	The first reclassification of IPC codes now complete in INPADOC
NEWS 10	JUN 26	TULSA/TULSA2 reloaded and enhanced with new search and and display fields
NEWS 11	JUN 28	Price changes in full-text patent databases EPFULL and PCTFULL
NEWS 12	JUL 11	CHEMSAFE reloaded and enhanced
NEWS 13	JUL 14	FSTA enhanced with Japanese patents
NEWS 14	JUL 19	Coverage of Research Disclosure reinstated in DWPI
NEWS 15	AUG 09	INSPEC enhanced with 1898-1968 archive
NEWS 16	AUG 28	ADISCTI Reloaded and Enhanced
NEWS 17	AUG 30	CA(SM)/CAPLUS(SM) Austrian patent law changes
NEWS 18	SEP 11	CA/CAPLUS enhanced with more pre-1907 records
NEWS 19	SEP 21	CA/CAPLUS fields enhanced with simultaneous left and right truncation
NEWS 20	SEP 25	CA(SM)/CAPLUS(SM) display of CA Lexicon enhanced
NEWS 21	SEP 25	CAS REGISTRY(SM) no longer includes Concord 3D coordinates
NEWS 22	SEP 25	CAS REGISTRY(SM) updated with amino acid codes for pyrrolysine
NEWS 23	SEP 28	CEABA-VTB classification code fields reloaded with new classification scheme
NEWS 24	OCT 02	MARPAT(R) now updated daily
NEWS EXPRESS	JUNE 30	CURRENT WINDOWS VERSION IS V8.01b, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006.
NEWS HOURS		STN Operating Hours Plus Help Desk Availability
NEWS LOGIN		Welcome Banner and News Items
NEWS IPC8		For general information regarding STN implementation of IPC 8
NEWS X25		X.25 communication option no longer available

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FILE 'HOME' ENTERED AT 17:51:52 ON 02 OCT 2006

=> file reg

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'REGISTRY' ENTERED AT 17:52:01 ON 02 OCT 2006

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DICTIONARY FILE UPDATES: 1 OCT 2006 HIGHEST RN 909214-11-5

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TSCA INFORMATION NOW CURRENT THROUGH June 30, 2006

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<http://www.cas.org/ONLINE/UG/regprops.html>

=> s akt or ? akt() oncogene? () protein? or akt () kinase () transform? () protein?

ADDITIONAL CHARACTERS REQUIRED AFTER '?' FOR LEFT TRUNCATION

Additional characters must follow the left truncation symbol in your search term. If your search term contains a punctuation mark before the truncation symbol and you are searching in a field that uses implied proximity, the system may interpret the truncation symbol as being at the beginning of a term. Implied proximity is used in search fields indexed as single words, for example, the Basic Index. To see which fields in the current file have left truncation, enter "HELP SFIELDS" at an arrow prompt (=>).

=> file hcaplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	1.32	1.53

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FILE COVERS 1907 - 2 Oct 2006 VOL 145 ISS 15  
FILE LAST UPDATED: 1 Oct 2006 (20061001/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s akt or ?akt () oncogene? () protein? or akt () kinase () transform? () protein?

11149 AKT  
18 AKTS  
11157 AKT  
(AKT OR AKTS)  
12944 ?AKT  
35156 ONCOGENE?  
2277690 PROTEIN?  
0 ?AKT (W) ONCOGENE? (W) PROTEIN?  
11149 AKT  
18 AKTS  
11157 AKT  
(AKT OR AKTS)  
273798 KINASE  
53395 KINASES  
282501 KINASE  
(KINASE OR KINASES)  
640626 TRANSFORM?  
2277690 PROTEIN?  
0 AKT (W) KINASE (W) TRANSFORM? (W) PROTEIN?  
L1 11157 AKT OR ?AKT (W) ONCOGENE? (W) PROTEIN? OR AKT (W) KINASE (W)  
TRANSFORM? (W) PROTEIN?

=> s l1 () inhibit?

1866618 INHIBIT?  
L2 383 L1 (W) INHIBIT?

=> s l2 and review/dt

1962466 REVIEW/DT  
L3 17 L2 AND REVIEW/DT

=> s l3 and cancer

292545 CANCER  
42708 CANCERS  
303633 CANCER  
(CANCER OR CANCERS)  
L4 8 L3 AND CANCER

=> d l4, ibib abs hitstr, 1-8

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L4 ANSWER 1 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2006:350627 HCAPLUS

DOCUMENT NUMBER: 144:465166

TITLE: Akt Signaling and Cancer: Surviving but not Moving On

AUTHOR(S): Toker, Alex; Yoeli-Lerner, Merav

CORPORATE SOURCE: Department of Pathology, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, USA

SOURCE: Cancer Research (2006), 66(8), 3963-3966

CODEN: CNREA8; ISSN: 0008-5472

PUBLISHER: American Association for Cancer Research

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. The frequent deregulation of the phosphoinositide 3-kinase/Akt survival signaling pathway in cancer has prompted significant interest in blocking this pathway to treat cancer. Recently, however, two studies have shown that the Akt isoform Akt1 limits the invasive migration of breast cancer cells. These studies suggest that Akt1 may have a dual role in tumorigenesis, acting not only pro-oncogenically by suppressing apoptosis but also anti-oncogenically by suppressing invasion and metastasis. We discuss the possible implications of these findings for therapeutic development of Akt inhibitors to treat cancer.

REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 2 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:1349832 HCAPLUS

DOCUMENT NUMBER: 144:307393

TITLE: Molecular strategies targeting the host component of cancer to enhance tumor response to radiation therapy

AUTHOR(S): Kim, Dong Wook; Huamani, Jessica; Fu, Allie; Hallahan, Dennis E.

CORPORATE SOURCE: Department of Radiation Oncology, Vanderbilt Ingram Cancer Center, Nashville, TN, USA

SOURCE: International Journal of Radiation Oncology, Biology, Physics (2005), Volume Date 2006, 64(1), 38-46

CODEN: IOBPD3; ISSN: 0360-3016

PUBLISHER: Elsevier Inc.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. The tumor microenvironment, in particular, the tumor vasculature, as an important target for the cytotoxic effects of radiation therapy is an established paradigm for cancer therapy. The authors review the evidence that the phosphoinositide 3-kinase (PI3K)/Akt pathway is activated in endothelial cells exposed to ionizing radiation (IR) and is a mol. target for the development of novel radiation sensitizing agents. On the basis of this premise, several promising preclin. studies that targeted the inhibition of the PI3K/Akt activation as a potential method of sensitizing the tumor vasculature to the cytotoxic effects of IR have been conducted. An innovative strategy to guide cytotoxic therapy in tumors treated with radiation and PI3K/Akt inhibitors is presented. The evidence supports a need for further investigation of combined-modality therapy that involves radiation therapy and inhibitors of PI3K/Akt pathway as a promising strategy for improving the treatment of patients with cancer.

REFERENCE COUNT: 68 THERE ARE 68 CITED REFERENCES AVAILABLE FOR THIS

## RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 3 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:1211155 HCAPLUS

DOCUMENT NUMBER: 144:16324

TITLE: The Akt/PKB pathway: molecular target for cancer drug discovery

AUTHOR(S): Cheng, Jin Q.; Lindsley, Craig W.; Cheng, George Z.; Yang, Hua; Nicosia, Santo V.

CORPORATE SOURCE: Departments of Pathology and Interdisciplinary Oncology, H Lee Moffitt Cancer Center and Research Institute, University of South Florida College of Medicine, Tampa, FL, 33612, USA

SOURCE: Oncogene (2005), 24(50), 7482-7492

CODEN: ONCNES; ISSN: 0950-9232

PUBLISHER: Nature Publishing Group

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. The serine/threonine kinase Akt/PKB pathway presents an exciting new target for mol. therapeutics, as it functions as a cardinal nodal point for transducing extracellular (growth factor and insulin) and intracellular (receptor tyrosine kinases, Ras and Src) oncogenic signals. In addition, alterations of the Akt pathway have been detected in a number of human malignancies. Ectopic expression of Akt, especially constitutively activated Akt, is sufficient to induce oncogenic transformation of cells and tumor formation in transgenic mice as well as chemoresistance. Akt has a wide range of downstream targets that regulate tumor-associated cell processes such as cell growth, cell cycle progression, survival, migration, epithelial-mesenchymal transition and angiogenesis. Blockage of Akt signaling results in apoptosis and growth inhibition of tumor cells with elevated Akt. The observed dependence of certain tumors on Akt signaling for survival and growth has wide implications for cancer therapy, offering the potential for preferential tumor cell killing. In the last several years, through combinatorial chemical, high-throughput and virtual screening, and traditional medicinal chemical, a number of inhibitors of

the Akt pathway have been identified. This review focuses on ongoing translational efforts to therapeutically target the Akt pathway.

REFERENCE COUNT: 131 THERE ARE 131 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 4 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:493934 HCAPLUS

DOCUMENT NUMBER: 143:242080

TITLE: Effects on cell viability

AUTHOR(S): Guzman, M.

CORPORATE SOURCE: Department of Biochemistry and Molecular Biology I, School of Biology, Complutense University, Madrid, 28040, Spain

SOURCE: Handbook of Experimental Pharmacology (2005), 168(Cannabinoids), 627-642

CODEN: HEPHD2; ISSN: 0171-2004

PUBLISHER: Springer GmbH

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. Cannabinoids are known to control the cell survival/death decision, leading to different outcomes that depend on the nature of the target cell and its proliferative or differentiation status. Cannabinoids

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induce growth arrest or apoptosis in a number of transformed cells in culture. They do so by modulating key cell signaling pathways involved in the control of tumor cell fate. The best-characterized example is cannabinoid-induced apoptosis of glioma cells, which occurs via sustained ceramide accumulation, extracellular signal-regulated kinase activation, and Akt inhibition. In addition, cannabinoid administration inhibits the angiogenesis and slows the growth of different types of tumors in laboratory animals. By contrast, most of the exptl.

evidence

indicates that cannabinoids protect normal neurons and glial cells from apoptosis as induced by toxic insults such as glutamatergic overstimulation, ischemia, and oxidative damage. It is therefore very likely that cannabinoids regulate cell survival and cell death pathways differently in tumor and non-tumor cells. Regarding immune cells, cannabinoids affect proliferation and survival in a complex and still obscure manner that depends on the exptl. setting. The findings reviewed here might set the basis for the use of cannabinoids in the treatment of cancer and neurodegenerative diseases.

REFERENCE COUNT: 87 THERE ARE 87 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 5 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:703858 HCAPLUS

DOCUMENT NUMBER: 141:184489

TITLE: AKT: A potential target for thyroid cancer therapy

AUTHOR(S): Kada, Faiza; Saji, Motoyasu; Ringel, Matthew D.

CORPORATE SOURCE: The Washington Hospital Center/MedStar Research Institute, Washington, DC, USA

SOURCE: Current Drug Targets: Immune, Endocrine and Metabolic Disorders (2004), 4(3), 181-185  
CODEN: CDTIBT; ISSN: 1568-0088

PUBLISHER: Bentham Science Publishers Ltd.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. Thyroid cancer is a heterogeneous disorder characterized by gene mutations that activate signaling pathways, and also by abnormalities in tumor suppressor genes and cell cycle proteins. Activation of the Akt/PKB signaling pathway appears to be an important event in thyroid tumorigenesis and, perhaps, in tumor progression too. Akt is activated in Cowden's syndrome through inactivation of PTEN, a neg. regulator of Akt. Cowden's syndrome is an autosomal dominant multiorgan hamartoma syndrome characterized by benign and malignant thyroid tumors, breast cancers, and colon cancers. In addition, the Akt pathway appears to be activated in a significant proportion of sporadic thyroid cancers through activation of growth factor pathways by thyroid oncogenes and/or receptor overexpression. Disruption of PI3-kinase activity pharmacol. or disruption of Akt signaling using dominant neg. cDNA expression have demonstrated salutary effects on several cancer models in vitro. Therefore, Akt represents an attractive target for pharmaceutical development for a variety of malignancies, including thyroid cancer.

REFERENCE COUNT: 72 THERE ARE 72 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 6 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:604067 HCAPLUS

DOCUMENT NUMBER: 141:199325

TITLE: Hypothesis: cannabinoid therapy for the treatment of

Updated Search

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gliomas?  
AUTHOR(S): Velasco, Guillermo; Galve-Roperh, Ismael; Sanchez, Cristina; Blazquez, Cristina; Guzman, Manuel  
CORPORATE SOURCE: School of Biology, Department of Biochemistry and Molecular Biology I, Complutense University, Madrid, 28040, Spain  
SOURCE: Neuropharmacology (2004), 47(3), 315-323  
CODEN: NEPHBW; ISSN: 0028-3908  
PUBLISHER: Elsevier B.V.  
DOCUMENT TYPE: Journal; General Review  
LANGUAGE: English  
AB A review. Gliomas, in particular glioblastoma multiforme or grade IV astrocytoma, are the most frequent class of malignant primary brain tumors and one of the most aggressive forms of cancer. Current therapeutic strategies for the treatment of glioblastoma multiforme are usually ineffective or just palliative. During the last few years, several studies have shown that cannabinoids-the active components of the plant Cannabis sativa and their derivs.-slow the growth of different types of tumors, including gliomas, in laboratory animals. Cannabinoids induce apoptosis of glioma cells in culture via sustained ceramide accumulation, extracellular signal-regulated kinase activation and Akt inhibition. In addition, cannabinoid treatment inhibits angiogenesis of gliomas in vivo. Remarkably, cannabinoids kill glioma cells selectively and can protect non-transformed glial cells from death. These and other findings reviewed here might set the basis for a potential use of cannabinoids in the management of gliomas.  
REFERENCE COUNT: 68 THERE ARE 68 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 7 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:506589 HCAPLUS  
DOCUMENT NUMBER: 141:98885  
TITLE: The development of phosphatidylinositol ether lipid analogues as inhibitors of the serine/threonine kinase, Akt  
AUTHOR(S): Gills, Joell J.; Dennis, Phillip A.  
CORPORATE SOURCE: NCI, Bethesda, MD, 20889, USA  
SOURCE: Expert Opinion on Investigational Drugs (2004), 13(7), 787-797  
CODEN: EOIDER; ISSN: 1354-3784  
PUBLISHER: Ashley Publications Ltd.  
DOCUMENT TYPE: Journal; General Review  
LANGUAGE: English  
AB A review. The serine/threonine kinase Akt is a component of the phosphatidylinositol 3'-kinase/Akt signal transduction pathway that is activated by receptor tyrosine kinases, activated Ras and integrins. As Akt regulates many processes crucial to carcinogenesis, and Akt activation has been observed in human cancers, intense efforts are underway to develop Akt inhibitors as cancer therapeutics. Towards this aim, phosphatidylinositol ether lipid analogs (PIAs), which are structurally similar to the products of phosphatidylinositol 3'-kinase, have been synthesized. PIAs inhibit Akt translocation, phosphorylation and kinase activity. Furthermore, they selectively induce apoptosis in cancer cell lines that depend on Akt for survival. This review will trace the development of PIAs, cover the biol. activities of PIAs and discuss future steps and challenges in their development.  
REFERENCE COUNT: 59 THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

Updated Search

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L4 ANSWER 8 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN  
ACCESSION NUMBER: 2002:607171 HCAPLUS  
DOCUMENT NUMBER: 138:162768  
TITLE: Targeting serine/threonine protein kinase B/Akt and  
cell-cycle checkpoint kinases for treating  
cancer  
AUTHOR(S): Li, Qun; Zhu, Gui-Dong  
CORPORATE SOURCE: Cancer Research, Global Pharmaceutical Discovery,  
Abbott Laboratories, Abbott Park, IL, 60064-6101, USA  
SOURCE: Current Topics in Medicinal Chemistry (Hilversum,  
Netherlands) (2002), 2(9), 939-971  
CODEN: CTMCCL; ISSN: 1568-0266  
PUBLISHER: Bentham Science Publishers Ltd.  
DOCUMENT TYPE: Journal; General Review  
LANGUAGE: English

AB A review. Over the past decade, protein kinases have emerged as a group of mol. targets with the potential to be "cancer-specific", allowing the selective targeting of cancer cells vs. normal cells. These selective anticancer drugs would eliminate the cytotoxic side effects that are associated with conventional cancer chemotherapy. This article will focus on two emerging and less-explored protein serine/threonine kinase targets: PKB/Akt and checkpoint kinase 1 (Chk1). Protein kinase B/Akts are a group of serine/threonine kinases that are overexpressed in a variety of human tumors. An Akt inhibitor would target the imbalance of pro-vs. anti-apoptosis regulation in cancerous as compared to healthy cells. Thus, a greater therapeutic window than conventional cytotoxic chemotherapy is expected. Cell-cycle checkpoints have become attractive targets since some of them, such as the G1/S checkpoint, are defective in most tumor cells. Inhibition of one or more of the remaining checkpoint(s) could make cancerous cells more sensitive than healthy cells toward DNA damaging agents or radiation therapy. Among the checkpoint kinases, Chk1 appears to be an attractive mol. target. Chk1 blocks the activation of the Cdc2-cyclin B kinase complex, and hence entry into mitosis, by disrupting the translocation of the phosphatase Cdc25C from the cytoplasm to the nucleus. A limited number of small mol. inhibitors in this emerging field and their mode of action will be reviewed.

REFERENCE COUNT: 275 THERE ARE 275 CITED REFERENCES AVAILABLE FOR  
THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE  
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(FILE 'HOME' ENTERED AT 17:51:52 ON 02 OCT 2006)

FILE 'REGISTRY' ENTERED AT 17:52:01 ON 02 OCT 2006

FILE 'HCAPLUS' ENTERED AT 17:54:03 ON 02 OCT 2006

L1 11157 S AKT OR ?AKT ( ) ONCOGENE? ( ) PROTEIN? OR AKT ( ) KINASE ( ) TRAN  
L2 383 S L1 ( ) INHIBIT?  
L3 17 S L2 AND REVIEW/DT  
L4 8 S L3 AND CANCER

=> s l3 not l4

L5 9 L3 NOT L4

=> s l5 and hyperinsulinisum?

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L6 0 HYPERINSULINISUM?  
0 L5 AND HYPERINSULINISUM?

=> s l4 and insulin?  
194576 INSULIN?

L7 1 L4 AND INSULIN?

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L7 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:1211155 HCAPLUS

DOCUMENT NUMBER: 144:16324

TITLE: The Akt/PKB pathway: molecular target for  
cancer drug discovery

AUTHOR(S): Cheng, Jin Q.; Lindsley, Craig W.; Cheng, George Z.;  
Yang, Hua; Nicosia, Santo V.

CORPORATE SOURCE: Departments of Pathology and Interdisciplinary  
Oncology, H Lee Moffitt Cancer Center and Research  
Institute, University of South Florida College of  
Medicine, Tampa, FL, 33612, USA

SOURCE: Oncogene (2005), 24(50), 7482-7492

CODEN: ONCNES; ISSN: 0950-9232

PUBLISHER: Nature Publishing Group

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. The serine/threonine kinase Akt/PKB pathway presents an exciting new target for mol. therapeutics, as it functions as a cardinal nodal point for transducing extracellular (growth factor and insulin) and intracellular (receptor tyrosine kinases, Ras and Src) oncogenic signals. In addition, alterations of the Akt pathway have been detected in a number of human malignancies. Ectopic expression of Akt, especially constitutively activated Akt, is sufficient to induce oncogenic transformation of cells and tumor formation in transgenic mice as well as chemoresistance. Akt has a wide range of downstream targets that regulate tumor-associated cell processes such as cell growth, cell cycle progression, survival, migration, epithelial-mesenchymal transition and angiogenesis. Blockage of Akt signaling results in apoptosis and growth inhibition of tumor cells with elevated Akt. The observed dependence of certain tumors on Akt signaling for survival and growth has wide implications for cancer therapy, offering the potential for preferential tumor cell killing. In the last several years, through combinatorial chemical, high-throughput and virtual screening, and traditional medicinal chemical, a number of inhibitors of the Akt pathway have been identified. This review focuses on ongoing translational efforts to therapeutically target the Akt pathway.

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L2 383 S L1 ( ) INHIBIT?

Updated Search

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L3 17 S L2 AND REVIEW/DT  
L4 8 S L3 AND CANCER  
L5 9 S L3 NOT L4  
L6 0 S L5 AND HYPERINSULINISUM?  
L7 1 S L4 AND INSULIN?

=> s l4 not l7  
L8 7 L4 NOT L7

=> s l5 not l7  
L9 9 L5 NOT L7

=> s l9 and hyperproliferative () disorder?  
1693 HYPERPROLIFERATIVE  
434770 DISORDER?  
442 HYPERPROLIFERATIVE (W) DISORDER?  
L10 0 L9 AND HYPERPROLIFERATIVE (W) DISORDER?

=> s l9 and angiogenesis?  
34518 ANGIOGENESIS?  
L11 1 L9 AND ANGIOGENESIS?

=> d l11, ibib abs, 1

L11 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2006 ACS on STN  
ACCESSION NUMBER: 2004:1003474 HCAPLUS  
DOCUMENT NUMBER: 142:168720  
TITLE: Canstatin, a endogenous inhibitor of  
angiogenesis and tumor growth  
AUTHOR(S): Su, Ying; Zhu, Jian-si  
CORPORATE SOURCE: Institute of Cancer Research, Nan Hua University,  
Hengyang, 421001, Peop. Rep. China  
SOURCE: Chinese Journal of Cancer Research (2004), 16(3),  
229-234  
CODEN: CJCRFH; ISSN: 1000-9604  
PUBLISHER: Chinese Journal of Cancer Research  
DOCUMENT TYPE: Journal; General Review  
LANGUAGE: English  
AB A review. Canstatin is a novel inhibitor of angiogenesis and  
tumor growth, derived from the C-terminal globular non-collageneous (NCI)  
domain of the  $\alpha 2$  chain of type IV collagen. It inhibits endothelial  
cell proliferation and migration in a dose-dependent manner, and induces  
endothelial cell apoptosis. In vivo expts. show that canstatin  
significantly inhibits solid tumor growth. The canstatin mediated  
inhibition of tumor is related to apoptosis. Canstatin- induced apoptosis  
is associated with phosphatidylinositol 3-kinase/Akt  
inhibition and is dependent upon signaling events transduced  
through membrane death receptor.  
REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS  
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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(FILE 'HOME' ENTERED AT 17:51:52 ON 02 OCT 2006)

FILE 'REGISTRY' ENTERED AT 17:52:01 ON 02 OCT 2006

FILE 'HCAPLUS' ENTERED AT 17:54:03 ON 02 OCT 2006

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L1 11157 S AKT OR ?AKT ( ) ONCOGENE? ( ) PROTEIN? OR AKT ( ) KINASE ( ) TRAN  
L2 383 S L1 ( ) INHIBIT?  
L3 17 S L2 AND REVIEW/DT  
L4 8 S L3 AND CANCER  
L5 9 S L3 NOT L4  
L6 0 S L5 AND HYPERINSULINISUM?  
L7 1 S L4 AND INSULIN?  
L8 7 S L4 NOT L7  
L9 9 S L5 NOT L7  
L10 0 S L9 AND HYPERPROLIFERATIVE ( ) DISORDER?  
L11 1 S L9 AND ANGIOGENESIS?

=> s l9 not l11

L12 8 L9 NOT L11

=> s l12 and restenosis?

7776 RESTENOSIS?

L13 0 L12 AND RESTENOSIS?

=> s l12 and inflammation?

159606 INFLAMMATION?

L14 0 L12 AND INFLAMMATION?

=> s l12 and autoimmune?

48030 AUTOIMMUNE?

L15 0 L12 AND AUTOIMMUNE?

=> s l12 and allergy?

44675 ALLERGY?

L16 0 L12 AND ALLERGY?

=> s l12 and asthma?

33692 ASTHMA?

L17 0 L12 AND ASTHMA?

=> d l12, ibib abs hitstr, 1-8

L12 ANSWER 1 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2006:488600 HCAPLUS

TITLE: Phosphoinositide 3-kinase/Akt signaling pathway and  
its therapeutical implications for human acute myeloid  
leukemia

AUTHOR(S): Martelli, A. M.; Nyakern, M.; Tabellini, G.; Bortul,  
R.; Tazzari, P. L.; Evangelisti, C.; Cocco, L.

CORPORATE SOURCE: Cell Signalling Laboratory, Dipartimento di Scienze  
Anatomiche Umane e Fisiopatologia dell'Apparato  
Locomotore, Sezione di Anatomia Umana, Universita di  
Bologna, Bologna, Italy

SOURCE: Leukemia (2006), 20(6), 911-928

CODEN: LEUKED; ISSN: 0887-6924

PUBLISHER: Nature Publishing Group

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB The phosphoinositide 3-kinase (PI3K)/Akt signaling pathway is crucial to  
many aspects of cell growth, survival and apoptosis, and its constitutive  
activation has been implicated in the both the pathogenesis and the  
progression of a wide variety of neoplasias. Hence, this pathway is an  
attractive target for the development of novel anticancer strategies.  
Recent studies showed that PI3K/Akt signaling is frequently activated in

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acute myeloid leukemia (AML) patient blasts and strongly contributes to proliferation, survival and drug resistance of these cells. Upregulation of the PI3K/Akt network in AML may be due to several reasons, including FLT3, Ras or c-Kit mutations. Small mols. designed to selectively target key components of this signal transduction cascade induce apoptosis and/or markedly increase conventional drug sensitivity of AML blasts in vitro. Thus, inhibitory mols. are currently being developed for clin. use either as single agents or in combination with conventional therapies. However, the PI3K/Akt pathway is important for many physiol. cellular functions and, in particular, for insulin signaling, so that its blockade in vivo might cause severe systemic side effects. In this review, we summarize the existing knowledge about PI3K/Akt signaling in AML cells and we examine the rationale for targeting this fundamental signal transduction network by means of selective pharmacol. inhibitors.

REFERENCE COUNT: 250 THERE ARE 250 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 2 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:1234702 HCAPLUS

DOCUMENT NUMBER: 145:116355

TITLE: Deguelin as a chemopreventive agent in mouse lung tumorigenesis induced by tobacco smoke carcinogens

AUTHOR(S): Hecht, Stephen S.

CORPORATE SOURCE: The Cancer Center, University of Minnesota, Minn., MN, 55455, USA

SOURCE: Journal of the National Cancer Institute (2005), 97(22), 1634-1635

CODEN: JNCIEQ; ISSN: 0027-8874

PUBLISHER: Oxford University Press

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. The research of Lee et al. (2005) entitled "Chemopreventive effects of deguelin, a novel Akt inhibitor, on tobacco-induced lung tumorigenesis" is reviewed with commentary and refs. The study by these authors showed that in mouse models, deguelin decreased the expression of pAkt in lungs and inhibited lung tumorigenesis induced by the tobacco smoke carcinogen benzo[a]pyrene (BAP) and 4(-methylnitrosamino)-1-(3-pyridyl)-1-butanone (NKK). The effects were particularly striking considering the relatively low dose of deguelin (4 mg/kg, twice a day) used in the chemoprevention study. Deguelin was effective when administered at the same time as BAP plus NKK or when given after carcinogen administration. An innovative aspect of this study was the use of microcomputed tomog. image anal. to detect lung tumors in live mice.

REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:1068675 HCAPLUS

DOCUMENT NUMBER: 144:120596

TITLE: Accelerating lead development by microwave-enhanced medicinal chemistry

AUTHOR(S): Shipe, William D.; Wolkenberg, Scott E.; Lindsley, Craig W.

CORPORATE SOURCE: Department of Medicinal Chemistry, Merck Research Laboratories, West Point, PA, 19486, USA

SOURCE: Drug Discovery Today: Technologies (2005), 2(2), 155-161

CODEN: DDTTB5; ISSN: 1740-6749

URL: <http://www.sciencedirect.com/science/journal/17406749>

PUBLISHER: Elsevier B.V.

DOCUMENT TYPE: Journal; General Review; (online computer file)

LANGUAGE: English

AB A review. Microwave-assisted organic synthesis (MAOS) addresses the need for accelerated chemical synthesis by providing many advantages over classical thermal conditions. Microwave instruments produced by Biotage, CEM and Milestone enable chemical to be safely and reproducibly performed on various scales and in a parallel fashion. To illustrate the high utility of this technol. for lead development, our Akt kinase program will be described wherein MAOS played a pivotal role in the identification of isoenzyme-selective Akt inhibitors.

REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 4 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:329631 HCAPLUS

DOCUMENT NUMBER: 142:456149

TITLE: Inhibitors of  $\beta$ -amyloid-induced toxicity by modulating the Akt signaling pathway

AUTHOR(S): Nakagami, Yasuhiro

CORPORATE SOURCE: Biological Research Laboratories, Sankyo Co., Ltd., Tokyo, 140-8710, Japan

SOURCE: Drug News &amp; Perspectives (2004), 17(10), 655-660

CODEN: DNPEED; ISSN: 0214-0934

PUBLISHER: Prous Science

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. The Akt signaling pathway plays a crucial role in neuronal survival, leading to inhibition of apoptosis. Many stimulants including neurotrophins are reported to activate this pathway in preclin. studies; however, there are no drugs for neurodegenerative diseases adopting such a concept on the market so far. Among neurodegenerative diseases, Alzheimer's disease is the most common and characterized by senile plaques and neurofibrillary tangles, which consist of  $\beta$ -amyloid and hyperphosphorylated tau, resp. Recent studies suggest that activation of Akt inhibits toxicity of  $\beta$ -amyloid and formation of neurofibrillary tangles, leading to protection of neurons against apoptosis. This review discusses the possibility of treatment of Alzheimer's disease by activating the Akt signaling pathway.

REFERENCE COUNT: 59 THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 5 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:475523 HCAPLUS

DOCUMENT NUMBER: 142:195821

TITLE: The molecular mechanisms regulating the phosphorylation of the NADPH oxidase component p47phox by phosphoinositide 3-kinase

AUTHOR(S): Yamamori, Tohru; Inanami, Osamu; Nagahata, Hajime; Kuwabara, Mikiyori

CORPORATE SOURCE: Lab. Radiat. Biol., Dep. Environ. Veterinary Sci., Grad. Sch. Veterinary Med., Hokkaido Univ., Sapporo, 060-0818, Japan

SOURCE: Jui Seikagaku (2003), 40(2), 63-76

CODEN: JSUEBY; ISSN: 1345-921X

10554187

PUBLISHER: Jui Seikagakkai  
DOCUMENT TYPE: Journal; General Review  
LANGUAGE: Japanese

AB A review. Superoxide production by NADPH oxidase is essential for the bactericidal properties of phagocytes. Phosphorylation of p47phox, one of the cytosolic components of NADPH oxidase, is a crucial step of the oxidase activation. Some evidences suggest that phosphoinositide 3-kinase (PI3K) is involved in p47phox phosphorylation, but it has not been fully understood how PI3K regulates it. The aim of this study was to examine the mechanism underlying the PI3K-regulation of p47phox phosphorylation. Pharmacol. inhibition of PI3K attenuated both fMLP-stimulated p47phox phosphorylation and NADPH oxidase activity in HL-60 cells differentiated to a neutrophil-like phenotype. Although fMLP elicited Akt activation in a PI3K-dependent manner, an Akt inhibitor had no effect on the oxidase activity triggered by fMLP. In vitro kinase assay revealed that Akt was unable to catalyze p47phox phosphorylation. Interestingly, the activation of cPKC and PKC $\delta$  after fMLP stimulation was dependent on PI3K. Furthermore, PI3K inhibitors reduced the activation of phospholipase Cy2 without affecting tyrosine phosphorylation on it. These results suggest that PI3K regulates the phosphorylation of NADPH oxidase component p47phox by controlling diacylglycerol-dependent PKCs but not Akt.

L12 ANSWER 6 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:361502 HCAPLUS  
DOCUMENT NUMBER: 141:306789  
TITLE: Anti apoptotic proteins as targets of chemotherapy  
AUTHOR(S): Smitha, V. B.; Ruby, John Anto  
CORPORATE SOURCE: Division of Cancer Biology, Rajiv Gandhi Centre for Biotechnology, Thiruvananthapuram, 695014, India  
SOURCE: Amala Research Bulletin (2003), 23, 1-6  
CODEN: ARBMCS; ISSN: 0971-4987  
PUBLISHER: Amala Cancer Research Centre  
DOCUMENT TYPE: Journal; General Review  
LANGUAGE: English

AB A review focuses on different anti-apoptotic mols. over-expressed by various tumors and their role in regulating the effectiveness of antitumor chemotherapy. It describes NF- $\kappa$ B; Bcl-2; Akt; inhibitor of apoptosis; and heat shock protein.

REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 7 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2003:453335 HCAPLUS  
DOCUMENT NUMBER: 139:225909  
TITLE: Decisions on life and death: FOXO forkhead transcription factors are in command when PKB/Akt is off duty  
AUTHOR(S): Burgering, Boudewijn M. T.; Medema, Rene H.  
CORPORATE SOURCE: Department of Physiological Chemistry and Center for Biomedical Genetics, University Medical Center Utrecht, Neth.  
SOURCE: Journal of Leukocyte Biology (2003), 73(6), 689-701  
CODEN: JLBIE7; ISSN: 0741-5400  
PUBLISHER: Federation of American Societies for Experimental Biology  
DOCUMENT TYPE: Journal; General Review  
LANGUAGE: English

AB A review. Forkhead transcription factors of the FOXO family are important

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downstream targets of protein kinase B (PKB)/Akt, a kinase shown to play a decisive role in cell proliferation and cell survival. Direct phosphorylation by PKB/Akt inhibits transcriptional activation by FOXO factors, causing their displacement from the nucleus into the cytoplasm. Work from recent years has shown that this family of transcription factors regulates the expression of a number of genes that are crucial for the proliferative status of a cell, as well as a number of genes involved in programmed cell death. As such, these transcription factors appear to play an essential role in many of the effects of PKB/Akt on cell proliferation and survival. Indeed, in cells of the hematopoietic system, mere activation of a FOXO factor is sufficient to activate a variety of proapoptotic genes and to trigger apoptosis. In contrast, in most other cell types, activation of FOXO blocks cellular proliferation and drives cells into a quiescent state. In such cell types, FOXO factors also provide the protective mechanisms that are required to adapt to the altered metabolic state of quiescent cells. Thus, as PKB/Akt signaling is switched off, FOXO factors take over to determine the fate of a cell, long-term survival in a quiescent state, or programmed cell death. This review summarizes our current understanding of the mechanisms by which PKB/Akt and FOXO factors regulate these decisions.

REFERENCE COUNT: 101 THERE ARE 101 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 8 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2003:446535 HCAPLUS

DOCUMENT NUMBER: 139:63860

TITLE: Akt inhibits DNA damage by suppressing p73, p53, Forkhead or all three?

AUTHOR(S): Basu, Subham

CORPORATE SOURCE: Signal Transduction Lab., Cancer Res., UK

SOURCE: Cell Cycle (2003), 2(2), 69-70

CODEN: CCEYAS; ISSN: 1538-4101

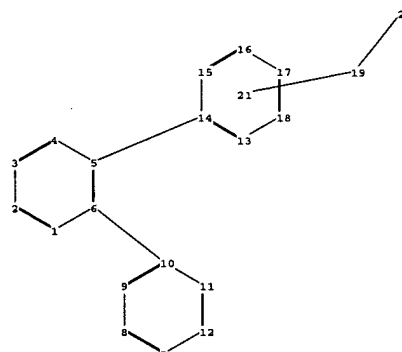
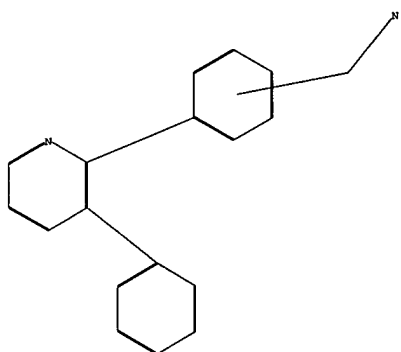
PUBLISHER: Landes Bioscience

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review discusses Akt regulation of DNA damage by suppression of transcription through p73, p53 and Forkhead. Recent data suggest that Akt regulation of p73-dependent DNA-damage is mediated primarily through YAP (Yes-associated protein), while Akt regulation of p53-dependent cell death is mediated through MDM2. Depending on the nature of the pro-apoptotic stimuli, DNA damaging or otherwise, the various Akt targets are possibly not only differentially regulated, as demonstrated by p53 and p73, but the same target may signal contrary effects, as seems to be the case for Forkhead. In an actual in vivo scenario, such as in a transformed cell that has evaded apoptosis by increasing Akt activity, the overall picture is probably generated by the coordinated regulation of a number of substrates, including but not limited to p53, p73 and Forkhead transcription factors.

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT



chain nodes :

19 20

ring nodes :

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18

chain bonds :

5-14 6-10 19-20

ring bonds :

1-2 1-6 2-3 3-4 4-5 5-6 7-8 7-12 8-9 9-10 10-11 11-12 13-14 13-18 14-15 15-16 16-17  
17-18

exact/norm bonds :

19-20

exact bonds :

5-14 6-10

normalized bonds :

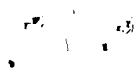
1-2 1-6 2-3 3-4 4-5 5-6 7-8 7-12 8-9 9-10 10-11 11-12 13-14 13-18 14-15 15-16 16-17  
17-18

isolated ring systems :

containing 1 : 7 : 13 :

Match level :

1:Atom 2:Atom 3:Atom 4:Atom 5:Atom 6:Atom 7:Atom 8:Atom 9:Atom 10:Atom 11:Atom 12:Atom  
13:Atom 14:Atom 15:Atom 16:Atom 17:Atom 18:Atom 19:CLASS20:CLASS21:Atom



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NEWS	3	FEB 27	New STN AnaVist pricing effective March 1, 2006
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NEWS	5	MAY 11	KOREAPAT updates resume
NEWS	6	MAY 19	Derwent World Patents Index to be reloaded and enhanced
NEWS	7	MAY 30	IPC 8 Rolled-up Core codes added to CA/CAPLUS and USPATFULL/USPAT2
NEWS	8	MAY 30	The F-Term thesaurus is now available in CA/CAPLUS
NEWS	9	JUN 02	The first reclassification of IPC codes now complete in INPADOC
NEWS	10	JUN 26	TULSA/TULSA2 reloaded and enhanced with new search and and display fields
NEWS	11	JUN 28	Price changes in full-text patent databases EPFULL and PCTFULL
NEWS	12	JUL 11	CHEMSAFE reloaded and enhanced
NEWS	13	JUL 14	FSTA enhanced with Japanese patents
NEWS	14	JUL 19	Coverage of Research Disclosure reinstated in DWPI
NEWS	15	AUG 09	INSPEC enhanced with 1898-1968 archive
NEWS	16	AUG 28	ADISCTI Reloaded and Enhanced
NEWS	17	AUG 30	CA(SM)/CAPLUS(SM) Austrian patent law changes
NEWS	18	SEP 11	CA/CAPLUS enhanced with more pre-1907 records
NEWS	19	SEP 21	CA/CAPLUS fields enhanced with simultaneous left and right truncation
NEWS	20	SEP 25	CA(SM)/CAPLUS(SM) display of CA Lexicon enhanced
NEWS	21	SEP 25	CAS REGISTRY(SM) no longer includes Concord 3D coordinates
NEWS	22	SEP 25	CAS REGISTRY(SM) updated with amino acid codes for pyrrolysine
NEWS	23	SEP 28	CEABA-VTB classification code fields reloaded with new classification scheme
NEWS	24	OCT 02	MARPAT(R) now updated daily
NEWS EXPRESS		JUNE 30	CURRENT WINDOWS VERSION IS V8.01b, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006.
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NEWS LOGIN			Welcome Banner and News Items
NEWS IPC8			For general information regarding STN implementation of IPC 8
NEWS X25			X.25 communication option no longer available

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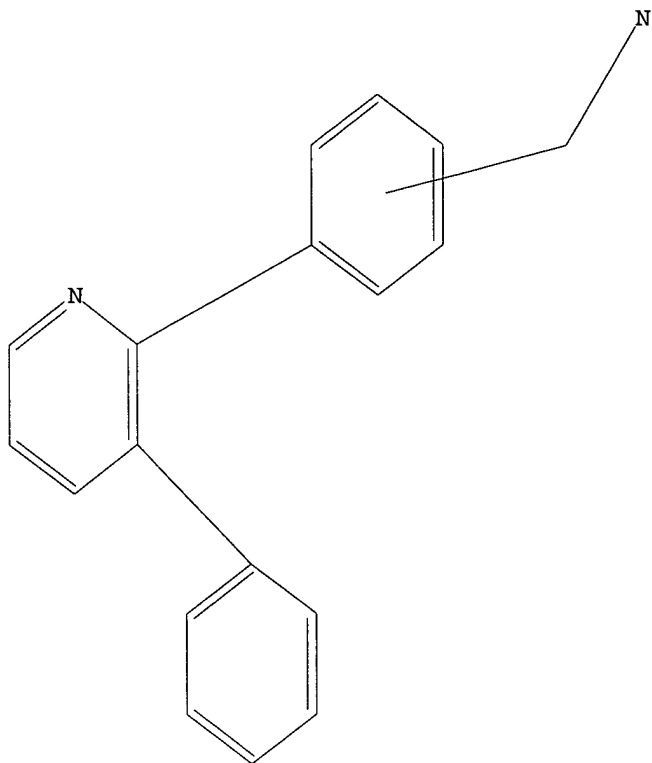
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100.0% PROCESSED 402 ITERATIONS  
SEARCH TIME: 00.00.01

1 ANSWERS

FULL FILE PROJECTIONS: ONLINE \*\*COMPLETE\*\*  
BATCH \*\*COMPLETE\*\*

PROJECTED ITERATIONS: 6838 TO 9242  
PROJECTED ANSWERS: 1 TO 80

L2 1 SEA SSS SAM L1

=> s l1 full

THE ESTIMATED SEARCH COST FOR FILE 'REGISTRY' IS 166.50 U.S. DOLLARS

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FULL SEARCH INITIATED 14:41:06 FILE 'REGISTRY'

FULL SCREEN SEARCH COMPLETED - 8595 TO ITERATE

100.0% PROCESSED 8595 ITERATIONS  
SEARCH TIME: 00.00.01

36 ANSWERS

L3 36 SEA SSS FUL L1

=> file hcaplus

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TOTAL

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4 L3

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284 DUGGAN, M?/AU

L6 2 L5 AND DUGGAN, M?/AU

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L6 ANSWER 1 OF 2 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:86368 HCAPLUS

DOCUMENT NUMBER: 142:211437

TITLE: Discovery of 2,3,5-trisubstituted pyridine derivatives as potent Akt1 and Akt2 dual inhibitors

AUTHOR(S): Zhao, Zhijian; Leister, William H.; Robinson, Ronald G.; Barnett, Stanley F.; Defeo-Jones, Deborah; Jones, Raymond E.; Hartman, George D.; Huff, Joel R.; Huber, Hans E.; Duggan, Mark E.; Lindsley, Craig W.

CORPORATE SOURCE: Department of Medicinal Chemistry, Technology Enabled Synthesis Group, Merck Research Laboratories, Merck & Co., West Point, PA, 19486, USA

SOURCE: Bioorganic & Medicinal Chemistry Letters (2005), 15(4), 905-909

CODEN: BMCLE8; ISSN: 0960-894X

PUBLISHER: Elsevier B.V.

Updated Search

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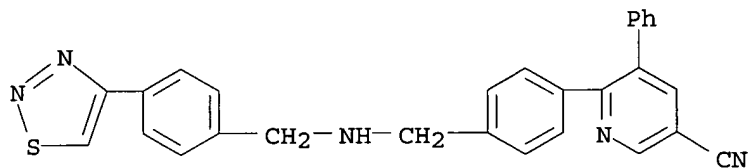
DOCUMENT TYPE: Journal  
LANGUAGE: English  
OTHER SOURCE(S): CASREACT 142:211437

AB This letter describes the discovery of a novel series of dual Akt1/Akt2 kinase inhibitors, based on a 2,3,5-trisubstituted pyridine scaffold. Compds. from this series, which contain a 5-tetrazolyl moiety, exhibit more potent inhibition of Akt2 than Akt1.

IT 790659-59-5P 790659-68-6P  
RL: PAC (Pharmacological activity); PKT (Pharmacokinetics); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study);  
PREP (Preparation); USES (Uses)  
(preparation of 2,3,5-trisubstituted pyridine derivs. as potent Akt1/Akt2 dual inhibitors)

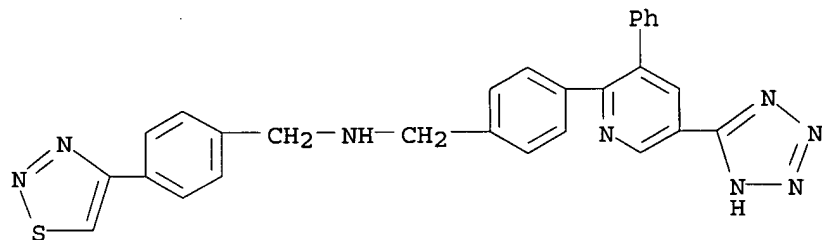
RN 790659-59-5 HCAPLUS

CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[4-(1,2,3-thiadiazol-4-yl)phenyl]methyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)



RN 790659-68-6 HCAPLUS

CN Benzenemethanamine, N-[[4-[3-phenyl-5-(1H-tetrazol-5-yl)-2-pyridinyl]phenyl]methyl]-4-(1,2,3-thiadiazol-4-yl)- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:964999 HCAPLUS

DOCUMENT NUMBER: 141:406038

TITLE: Substituted pyridine compounds as inhibitors of protein kinase Akt activity for treating cancer

INVENTOR(S): Duggan, Mark E.; Lindsley, Craig W.; Wu, Zhicai; Zhao, Zhijian; Hartnett, John C.

PATENT ASSIGNEE(S): Merck & Co., Inc., USA

SOURCE: PCT Int. Appl., 58 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

Updated Search

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004096135	A2	20041111	WO 2004-US12265	20040420
WO 2004096135	A3	20050324		
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AU 2004233835	A1	20041111	AU 2004-233835	20040420
CA 2522435	AA	20041111	CA 2004-2522435	20040420
EP 1631548	A2	20060308	EP 2004-750420	20040420
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK				
CN 1809536	A	20060726	CN 2004-80017036	20040420
PRIORITY APPLN. INFO.:			US 2003-465125P	P 20030424
			WO 2004-US12265	W 20040420

OTHER SOURCE(S): MARPAT 141:406038

AB The present invention is directed to compds. which contain a substituted pyridine moiety which inhibit the activity of Akt, a serine/threonine protein kinase. The invention is further directed to chemotherapeutic compns. containing the compds. of this invention and methods for treating cancer comprising administration of the compds. of the invention.

IT 790659-74-4P

RL: BSU (Biological study, unclassified); PAC (Pharmacological activity); RCT (Reactant); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); RACT (Reactant or reagent); USES (Uses)

(substituted pyridine compds. as inhibitors of protein kinase Akt activity for treating cancer)

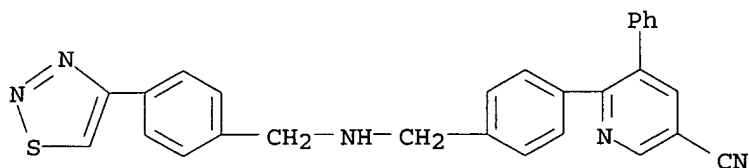
RN 790659-74-4 HCAPLUS

CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[4-(1,2,3-thiadiazol-4-yl)phenyl]methyl]amino]methyl]phenyl]-, trifluoroacetate (9CI) (CA INDEX NAME)

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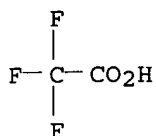
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CM 2

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CRN 76-05-1  
CMF C2 H F3 O2

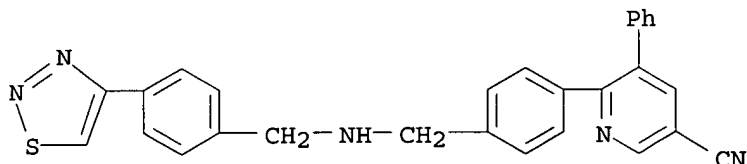


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790659-68-6P 790659-69-7P 790659-70-0P  
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790659-75-5P 790659-76-6P 790659-77-7P  
790659-78-8P 790659-79-9P 790659-80-2P  
790659-81-3P 790659-82-4P 790659-83-5P  
790659-84-6P

RL: BSU (Biological study, unclassified); PAC (Pharmacological activity);  
SPN (Synthetic preparation); THU (Therapeutic use); BIOL  
(Biological study); PREP (Preparation); USES (Uses)  
(substituted pyridine compds. as inhibitors of protein kinase Akt  
activity for treating cancer)

RN 790659-59-5 HCAPLUS

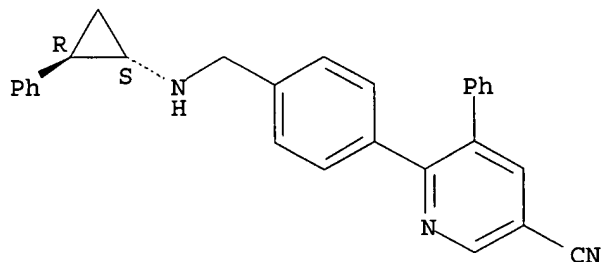
CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[4-(1,2,3-thiadiazol-4-yl)phenyl]methyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)



RN 790659-60-8 HCAPLUS

CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[(1S,2R)-2-phenylcyclopropyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

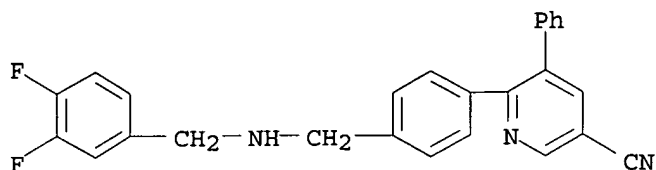


RN 790659-61-9 HCAPLUS

CN 3-Pyridinecarbonitrile, 6-[4-[[[(3,4-difluorophenyl)methyl]amino]methyl]phenyl]-5-phenyl- (9CI) (CA INDEX NAME)

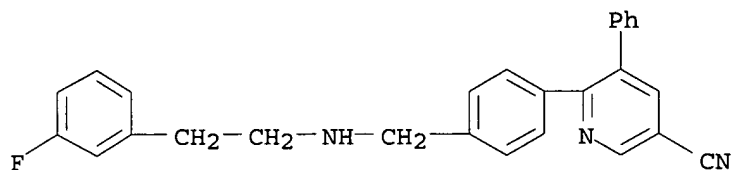
Updated Search

10554187



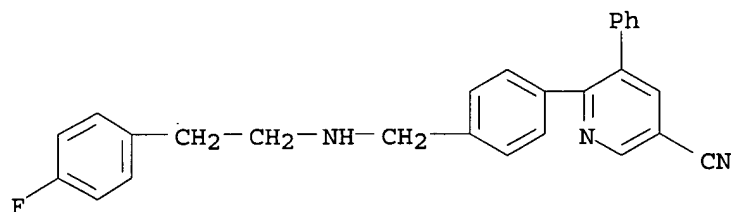
RN 790659-62-0 HCAPLUS

CN 3-Pyridinecarbonitrile, 6-[4-[[[2-(3-fluorophenyl)ethyl]amino]methyl]phenyl]-5-phenyl- (9CI) (CA INDEX NAME)



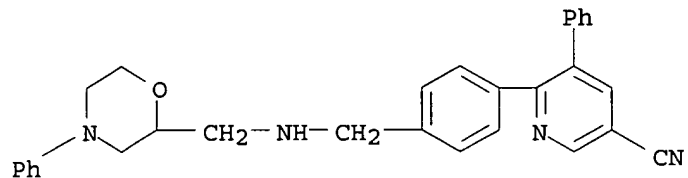
RN 790659-63-1 HCAPLUS

CN 3-Pyridinecarbonitrile, 6-[4-[[[2-(4-fluorophenyl)ethyl]amino]methyl]phenyl]-5-phenyl- (9CI) (CA INDEX NAME)



RN 790659-64-2 HCAPLUS

CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[4-(phenylmethyl)-2-morpholinyl]methyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)

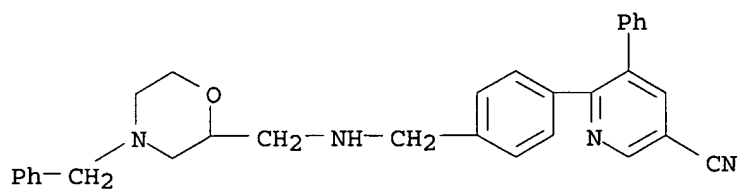


RN 790659-65-3 HCAPLUS

CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[4-(phenylmethyl)-2-morpholinyl]methyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)

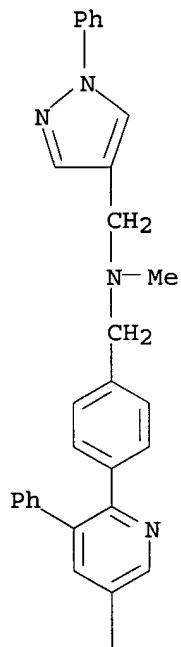
Updated Search

10554187



RN 790659-66-4 HCAPLUS  
CN 3-Pyridinecarbonitrile, 6-[4-[[methyl[(1-phenyl-1H-pyrazol-4-yl)methyl]amino]methyl]phenyl]-5-phenyl- (9CI) (CA INDEX NAME)

PAGE 1-A



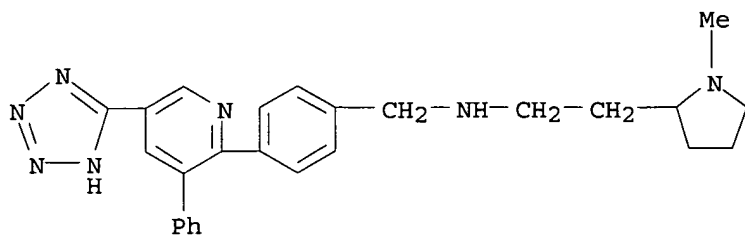
PAGE 2-A



RN 790659-67-5 HCAPLUS  
CN 2-Pyrrolidineethanamine, 1-methyl-N-[[4-[3-phenyl-5-(1H-tetrazol-5-yl)-2-pyridinyl]phenyl]methyl]- (9CI) (CA INDEX NAME)

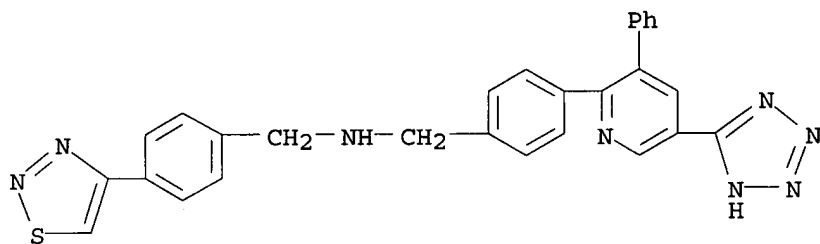
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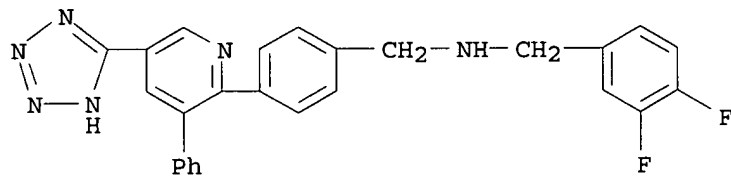
RN 790659-68-6 HCAPLUS

CN Benzenemethanamine, N-[[4-[3-phenyl-5-(1H-tetrazol-5-yl)-2-pyridinyl]phenyl]methyl]-4-(1,2,3-thiadiazol-4-yl)- (9CI) (CA INDEX NAME)



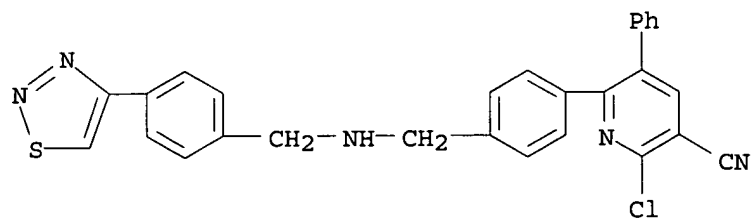
RN 790659-69-7 HCAPLUS

CN Benzenemethanamine, 3,4-difluoro-N-[[4-[3-phenyl-5-(1H-tetrazol-5-yl)-2-pyridinyl]phenyl]methyl]- (9CI) (CA INDEX NAME)



RN 790659-70-0 HCAPLUS

CN 3-Pyridinecarbonitrile, 2-chloro-5-phenyl-6-[4-[[[4-(1,2,3-thiadiazol-4-yl)phenyl]methyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)

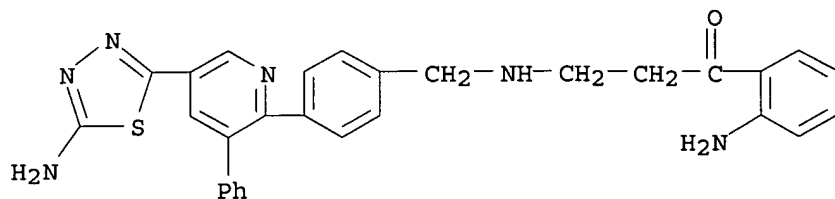


RN 790659-71-1 HCAPLUS

CN 1-Propanone, 1-(2-aminophenyl)-3-[[[4-[5-(5-amino-1,3,4-thiadiazol-2-yl)-3-phenyl-2-pyridinyl]phenyl]methyl]amino]- (9CI) (CA INDEX NAME)

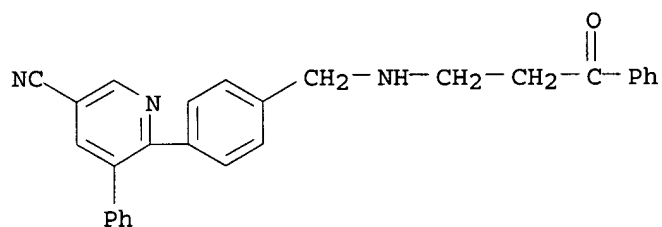
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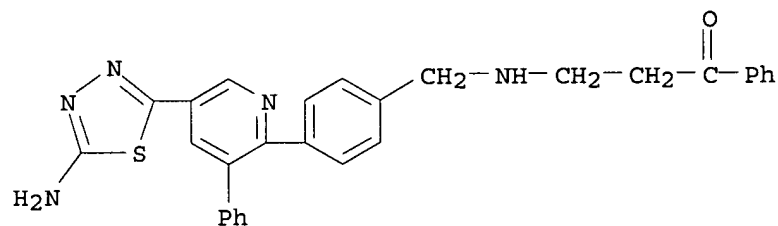
RN 790659-72-2 HCAPLUS

CN 3-Pyridinecarbonitrile, 6-[4-[[3-oxo-3-phenylpropyl]amino]methyl]phenyl]-5-phenyl- (9CI) (CA INDEX NAME)



RN 790659-73-3 HCAPLUS

CN 1-Propanone, 3-[[[4-[5-(5-amino-1,3,4-thiadiazol-2-yl)-3-phenyl-2-pyridinyl]phenyl]methyl]amino]-1-phenyl- (9CI) (CA INDEX NAME)



RN 790659-75-5 HCAPLUS

CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[(1S,2R)-2-phenylcyclopropyl]amino]methyl]phenyl]-, trifluoroacetate (9CI) (CA INDEX NAME)

CM 1

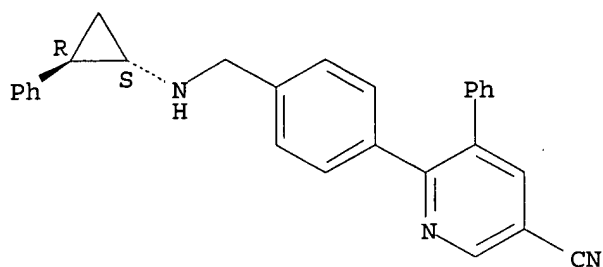
CRN 790659-60-8

CMF C28 H23 N3

Absolute stereochemistry.

Updated Search

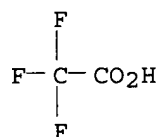
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CM 2

CRN 76-05-1

CMF C2 H F3 O2



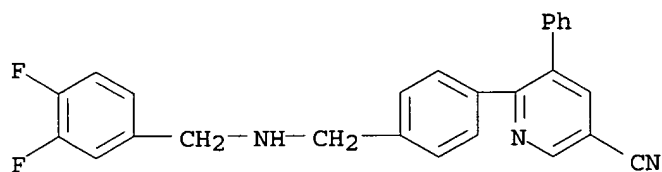
RN 790659-76-6 HCAPLUS

CN 3-Pyridinecarbonitrile, 6-[4-[[[(3,4-difluorophenyl)methyl]amino]methyl]phenyl]-5-phenyl-, trifluoroacetate (9CI) (CA INDEX NAME)

CM 1

CRN 790659-61-9

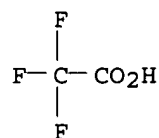
CMF C26 H19 F2 N3



CM 2

CRN 76-05-1

CMF C2 H F3 O2



RN 790659-77-7 HCAPLUS

Updated Search

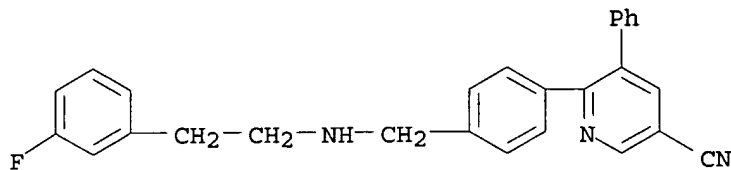
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CN 3-Pyridinecarbonitrile, 6-[4-[[[2-(3-fluorophenyl)ethyl]amino]methyl]phenyl]-5-phenyl-, trifluoroacetate (9CI) (CA INDEX NAME)

CM 1

CRN 790659-62-0

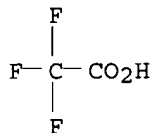
CMF C27 H22 F N3



CM 2

CRN 76-05-1

CMF C2 H F3 O2



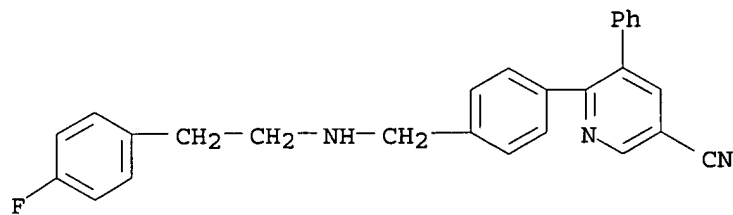
RN 790659-78-8 HCAPLUS

CN 3-Pyridinecarbonitrile, 6-[4-[[[2-(4-fluorophenyl)ethyl]amino]methyl]phenyl]-5-phenyl-, trifluoroacetate (9CI) (CA INDEX NAME)

CM 1

CRN 790659-63-1

CMF C27 H22 F N3



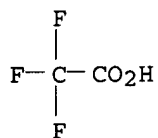
CM 2

CRN 76-05-1

CMF C2 H F3 O2

Updated Search

10554187



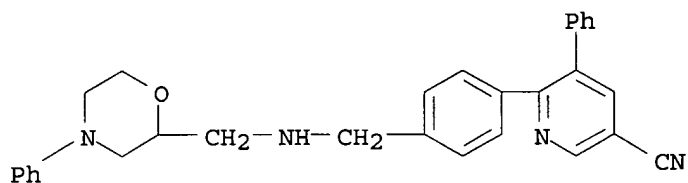
RN 790659-79-9 HCAPLUS

CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[(4-phenyl-2-morpholinyl)methyl]amino]methyl]phenyl]-, trifluoroacetate (9CI) (CA INDEX NAME)

CM 1

CRN 790659-64-2

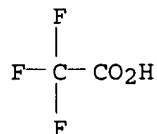
CMF C30 H28 N4 O



CM 2

CRN 76-05-1

CMF C2 H F3 O2



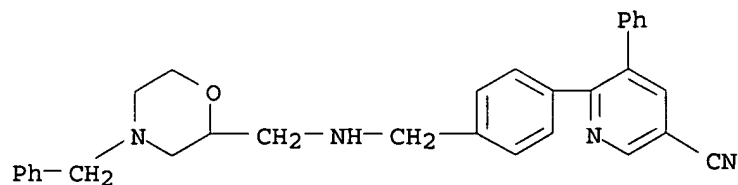
RN 790659-80-2 HCAPLUS

CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[4-(phenylmethyl)-2-morpholinyl]methyl]amino]methyl]phenyl]-, trifluoroacetate (9CI) (CA INDEX NAME)

CM 1

CRN 790659-65-3

CMF C31 H30 N4 O



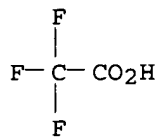
Updated Search

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CM 2

CRN 76-05-1

CMF C2 H F3 O2



RN 790659-81-3 HCAPLUS

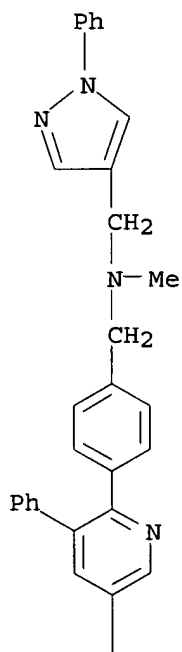
CN 3-Pyridinecarbonitrile, 6-[4-[[methyl[(1-phenyl-1H-pyrazol-4-yl)methyl]amino]methyl]phenyl]-5-phenyl-, trifluoroacetate (9CI) (CA INDEX NAME)

CM 1

CRN 790659-66-4

CMF C30 H25 N5

PAGE 1-A



PAGE 2-A

CN

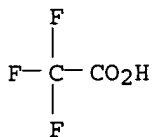
Updated Search

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CM 2

CRN 76-05-1

CMF C2 H F3 O2



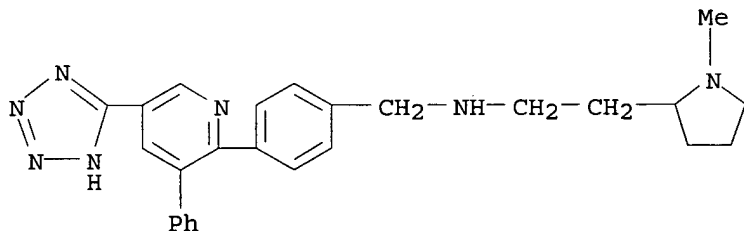
RN 790659-82-4 HCAPLUS

CN 2-Pyrrolidineethanamine, 1-methyl-N-[[4-[3-phenyl-5-(1H-tetrazol-5-yl)-2-pyridinyl]phenyl]methyl]-, trifluoroacetate (9CI) (CA INDEX NAME)

CM 1

CRN 790659-67-5

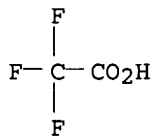
CMF C26 H29 N7



CM 2

CRN 76-05-1

CMF C2 H F3 O2



RN 790659-83-5 HCAPLUS

CN Benzenemethanamine, N-[[4-[3-phenyl-5-(1H-tetrazol-5-yl)-2-pyridinyl]phenyl]methyl]-4-(1,2,3-thiadiazol-4-yl)-, trifluoroacetate (9CI) (CA INDEX NAME)

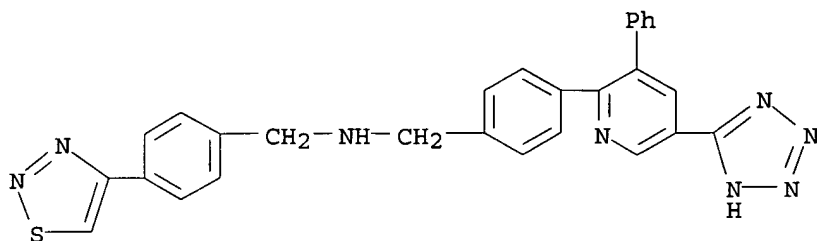
CM 1

CRN 790659-68-6

CMF C28 H22 N8 S

Updated Search

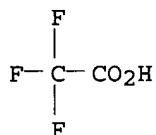
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CM 2

CRN 76-05-1

CMF C2 H F3 O2



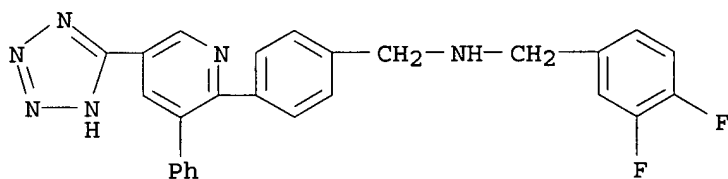
RN 790659-84-6 HCAPLUS

CN Benzenemethanamine, 3,4-difluoro-N-[[4-[3-phenyl-5-(1H-tetrazol-5-yl)-2-pyridinyl]phenyl]methyl]-, trifluoroacetate (9CI) (CA INDEX NAME)

CM 1

CRN 790659-69-7

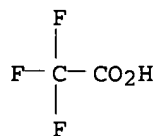
CMF C26 H20 F2 N6



CM 2

CRN 76-05-1

CMF C2 H F3 O2



IT 790659-59-5D, salts, stereoisomers 790659-60-8D, salts, stereoisomers 790659-61-9D, salts, stereoisomers

Updated Search

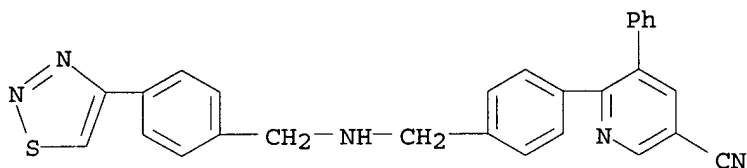
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790659-62-0D, salts, stereoisomers 790659-63-1D, salts,  
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790659-65-3D, salts, stereoisomers 790659-66-4D, salts,  
stereoisomers 790659-67-5D, salts, stereoisomers  
790659-68-6D, salts, stereoisomers 790659-69-7D, salts,  
stereoisomers 790659-70-0D, salts, stereoisomers  
790659-71-1D, salts, stereoisomers 790659-72-2D, salts,  
stereoisomers 790659-73-3D, salts, stereoisomers  
790659-85-7 790659-86-8

RL: BSU (Biological study, unclassified); PAC (Pharmacological activity);  
THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
(substituted pyridine compds. as inhibitors of protein kinase Akt  
activity for treating cancer)

RN 790659-59-5 HCAPLUS

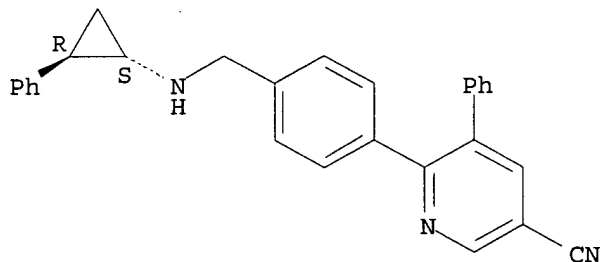
CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[4-(1,2,3-thiadiazol-4-yl)phenyl]methyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)



RN 790659-60-8 HCAPLUS

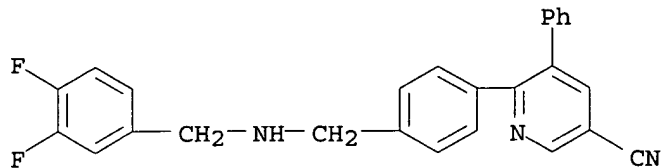
CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[(1S,2R)-2-phenylcyclopropyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



RN 790659-61-9 HCAPLUS

CN 3-Pyridinecarbonitrile, 6-[4-[[[(3,4-difluorophenyl)methyl]amino]methyl]phenyl]-5-phenyl- (9CI) (CA INDEX NAME)



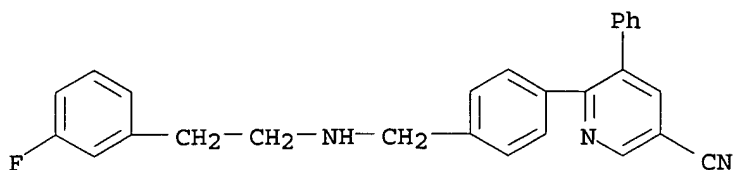
RN 790659-62-0 HCAPLUS

CN 3-Pyridinecarbonitrile, 6-[4-[[[2-(3-fluorophenyl)ethyl]amino]methyl]phenyl]-5-phenyl- (9CI) (CA INDEX NAME)

Updated Search

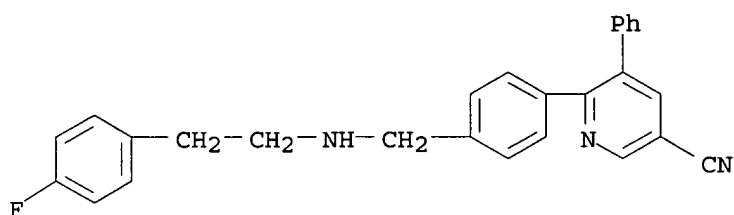
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1]-5-phenyl- (9CI) (CA INDEX NAME)



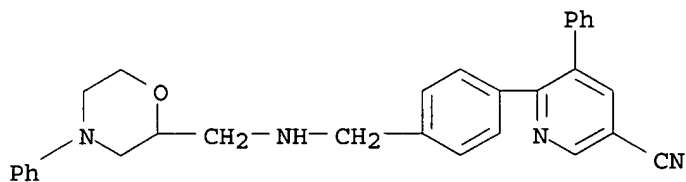
RN 790659-63-1 HCAPLUS

CN 3-Pyridinecarbonitrile, 6-[4-[[2-(4-fluorophenyl)ethyl]amino]methyl]phenyl]-5-phenyl- (9CI) (CA INDEX NAME)



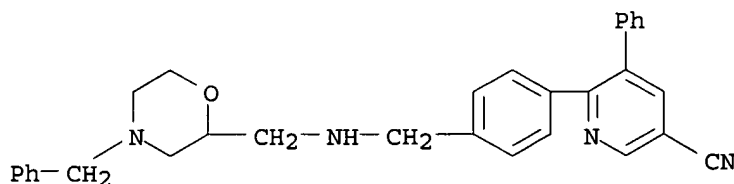
RN 790659-64-2 HCAPLUS

CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[4-phenyl-2-morpholinyl]methyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)



RN 790659-65-3 HCAPLUS

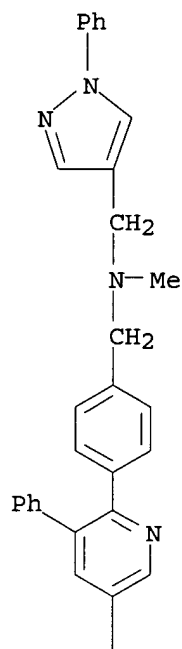
CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[4-(phenylmethyl)-2-morpholinyl]methyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)



RN 790659-66-4 HCAPLUS

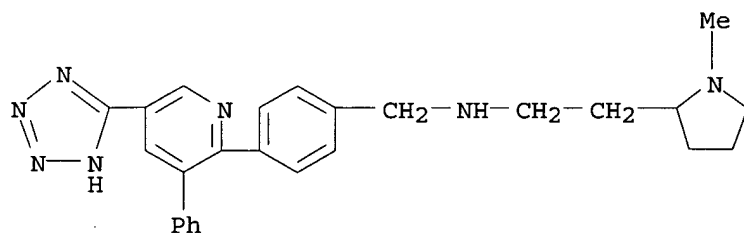
CN 3-Pyridinecarbonitrile, 6-[4-[methyl[(1-phenyl-1H-pyrazol-4-yl)methyl]amino]methyl]phenyl]-5-phenyl- (9CI) (CA INDEX NAME)

Updated Search



RN 790659-67-5 HCAPLUS

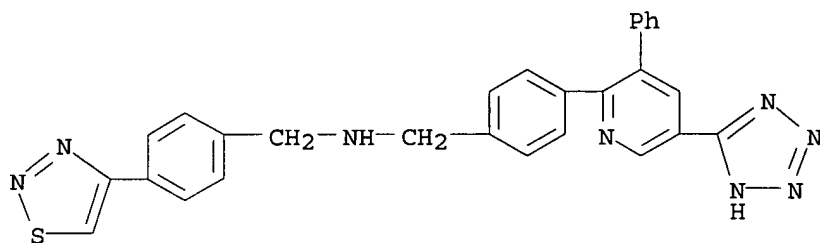
CN 2-Pyrrolidineethanamine, 1-methyl-N-[[4-[3-phenyl-5-(1H-tetrazol-5-yl)-2-pyridinyl]phenyl]methyl]-(9CI) (CA INDEX NAME)



RN 790659-68-6 HCAPLUS

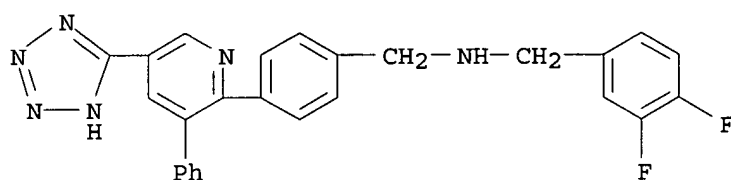
CN Benzenemethanamine, N-[[4-[3-phenyl-5-(1H-tetrazol-5-yl)-2-pyridinyl]phenyl]methyl]-4-(1,2,3-thiadiazol-4-yl)-(9CI) (CA INDEX NAME)

10554187



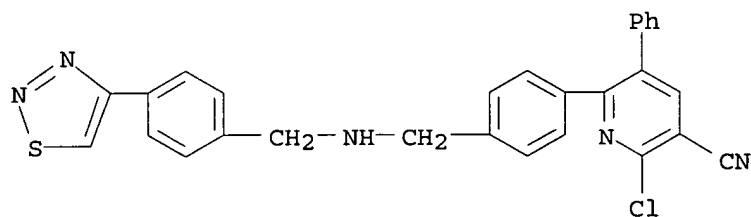
RN 790659-69-7 HCAPLUS

CN Benzenemethanamine, 3,4-difluoro-N-[[4-[3-phenyl-5-(1H-tetrazol-5-yl)-2-pyridinyl]phenyl]methyl]- (9CI) (CA INDEX NAME)



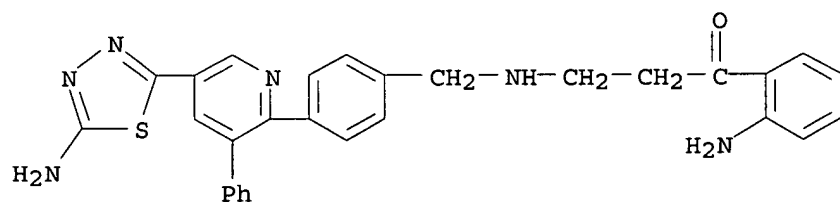
RN 790659-70-0 HCAPLUS

CN 3-Pyridinecarbonitrile, 2-chloro-5-phenyl-6-[[4-[[[4-(1,2,3-thiadiazol-4-yl)phenyl]methyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)



RN 790659-71-1 HCAPLUS

CN 1-Propanone, 1-(2-aminophenyl)-3-[[[4-[5-(5-amino-1,3,4-thiadiazol-2-yl)-3-phenyl-2-pyridinyl]phenyl]methyl]amino]- (9CI) (CA INDEX NAME)

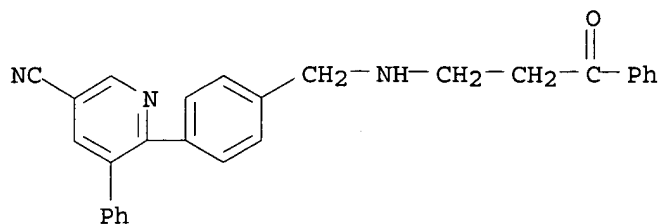


RN 790659-72-2 HCAPLUS

CN 3-Pyridinecarbonitrile, 6-[[4-[[[3-oxo-3-phenylpropyl]amino]methyl]phenyl]-5-phenyl]- (9CI) (CA INDEX NAME)

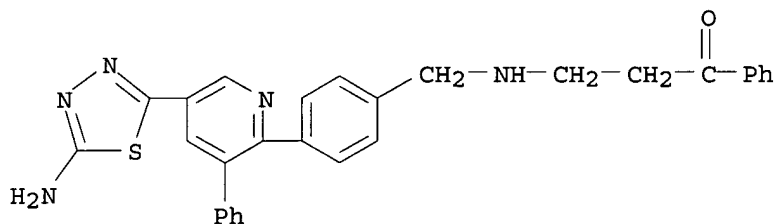
Updated Search

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RN 790659-73-3 HCAPLUS

CN 1-Propanone, 3-[[[4-[5-(5-amino-1,3,4-thiadiazol-2-yl)-3-phenyl-2-pyridinyl]phenyl]methyl]amino]-1-phenyl- (9CI) (CA INDEX NAME)



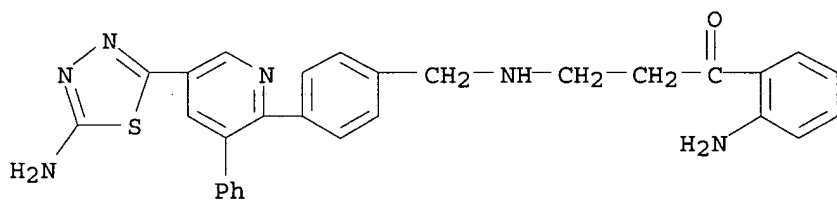
RN 790659-85-7 HCAPLUS

CN 1-Propanone, 1-(2-aminophenyl)-3-[[[4-[5-(5-amino-1,3,4-thiadiazol-2-yl)-3-phenyl-2-pyridinyl]phenyl]methyl]amino]-, trifluoroacetate (9CI) (CA INDEX NAME)

CM 1

CRN 790659-71-1

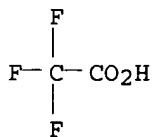
CMF C29 H26 N6 O S



CM 2

CRN 76-05-1

CMF C2 H F3 O2



Updated Search

10554187

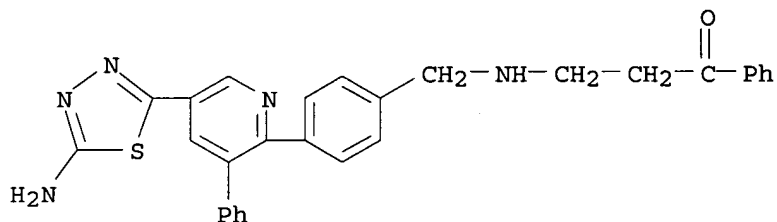
RN 790659-86-8 HCAPLUS

CN 1-Propanone, 3-[[[4-[5-(5-amino-1,3,4-thiadiazol-2-yl)-3-phenyl-2-pyridinyl]phenyl]methyl]amino]-1-phenyl-, trifluoroacetate (9CI) (CA INDEX NAME)

CM 1

CRN 790659-73-3

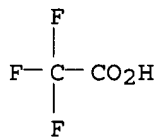
CMF C29 H25 N5 O S



CM 2

CRN 76-05-1

CMF C2 H F3 O2



=> d his

(FILE 'HOME' ENTERED AT 14:36:42 ON 02 OCT 2006)

FILE 'REGISTRY' ENTERED AT 14:39:04 ON 02 OCT 2006

L1 STRUCTURE UPLOADED

L2 1 S L1

L3 36 S L1 FULL

FILE 'HCAPLUS' ENTERED AT 14:41:10 ON 02 OCT 2006

L4 4 S L3

L5 4 S L3/THU

L6 2 S L5 AND DUGGAN, M?/AU

=> s l5 not l6

L7 2 L5 NOT L6

=> s l7 and lindsley, c?/au

100 LINDSLEY, C?/AU

L8 0 L7 AND LINDSLEY, C?/AU

=> s l7 and wu, z?/au

Updated Search

10554187

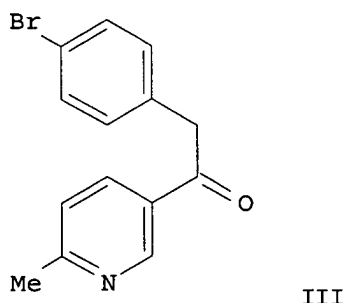
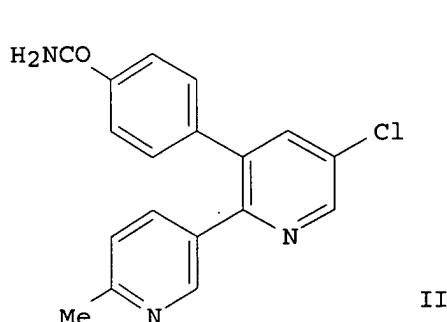
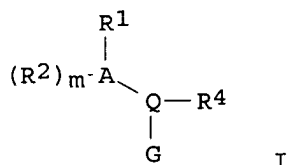
10714 WU, Z?/AU  
L9 0 L7 AND WU, Z?/AU  
  
=> s 17 and zhao, z?/au  
7927 ZHAO, Z?/AU  
L10 0 L7 AND ZHAO, Z?/AU  
  
=> s 17 and hartnett, j?/au  
222 HARTNETT, J?/AU  
L11 0 L7 AND HARTNETT, J?/AU  
  
=> d 17, ibib abs hitstr, 1-2

L7 ANSWER 1 OF 2 HCAPLUS COPYRIGHT 2006 ACS on STN  
ACCESSION NUMBER: 2005:1288063 HCAPLUS  
DOCUMENT NUMBER: 144:36364  
TITLE: Bicyclic compounds  
INVENTOR(S): Hirai, Miki; Kusama, Mari; Hosaka, Toshihiro; Kohnomi, Shuntarou  
PATENT ASSIGNEE(S): Tanabe Seiyaku Co., Ltd., Japan  
SOURCE: PCT Int. Appl., 68 pp.  
CODEN: PIXXD2  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 1  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005115984	A2	20051208	WO 2005-JP10287	20050530
WO 2005115984	A3	20060302		
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW			
RW:	BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			

PRIORITY APPLN. INFO.:  
JP 2004-160660 A 20040531  
JP 2004-191849 A 20040629  
US 2004-584142P P 20040701  
JP 2004-348136 A 20041201

OTHER SOURCE(S): MARPAT 144:36364  
GI



AB Heterocyclic compds. I [Q = pyridine or pyrimidine; A = benzene or heteroarom. ring; G = ring B optionally substituted with R<sub>3</sub>, or amino optionally substituted by one or two selected from the group consisting of alkyl, aralkyl and cycloalkyl; ring B = benzene, heterocyclic ring, cycloalkane or cycloalkene; R<sub>1</sub> = CON(R<sub>6</sub>)R<sub>5</sub>, CON(R<sub>6</sub>)OR<sub>5</sub>, CONHN(R<sub>6</sub>)R<sub>5</sub>, COON(R<sub>6</sub>)COR<sub>5</sub>, CON(R<sub>6</sub>)SO<sub>2</sub>R<sub>5</sub>, COR<sub>5</sub>, CO<sub>2</sub>R<sub>5</sub>, CN; R<sub>2</sub> and R<sub>3</sub> may be the same or different from each other, and each = CN, NO<sub>2</sub>, OH, alkoxy, halo, carboxyl, etc.; m = 0, 1 or 2; R<sub>4</sub> = H, CN, OH, halo, alkoxy, carbamoyl, etc.; R<sub>5</sub> and R<sub>6</sub> may be the same or different from each other, and each = H, an optionally substituted alkyl, cycloalkyl, aryl, heterocyclic, alkoxy carbonyl, or R<sub>5</sub> and R<sub>6</sub> may form an optionally substituted heterocyclic ring in combination with atoms to which they are bonded] and pharmaceutically acceptable salt were prepared as calcium-activated K channel opener useful for treatment of pollakiuria, urinary incontinence, chronic obstructive lung disease and prophylaxis. Thus, compound II was prepared via heterocyclization reaction of III with Vilsmeier agent, and showed a relaxation effect on K-induced contraction of isolated urinary bladder.

IT 870723-07-2P 870723-08-3P 870723-09-4P

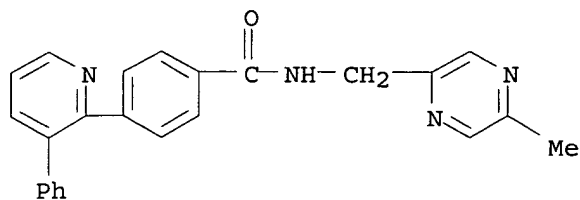
RL: PAC (Pharmacological activity); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)

(preparation of heterocyclic compds. as calcium-activated K channel opener for treatment of pollakiuria, urinary incontinence, chronic obstructive lung disease and prophylaxis)

RN 870723-07-2 HCAPLUS

CN Benzamide, N-[(5-methylpyrazinyl)methyl]-4-(3-phenyl-2-pyridinyl)-, monohydrochloride (9CI) (CA INDEX NAME)

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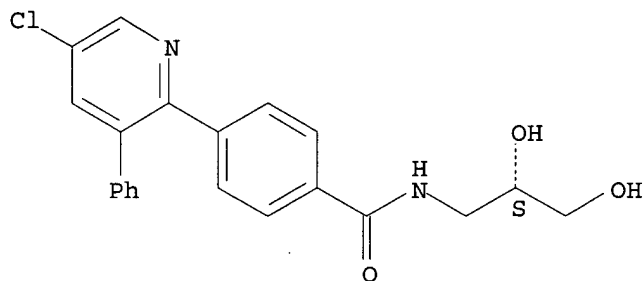


● HCl

RN 870723-08-3 HCAPLUS

CN Benzamide, 4-(5-chloro-3-phenyl-2-pyridinyl)-N-[(2S)-2,3-dihydroxypropyl]-, monohydrochloride (9CI) (CA INDEX NAME)

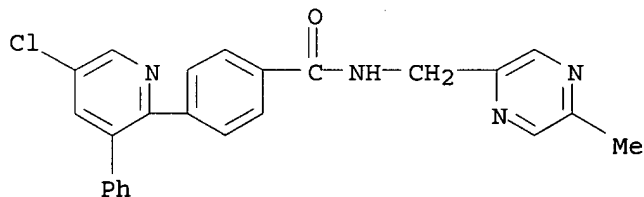
Absolute stereochemistry.



● HCl

RN 870723-09-4 HCAPLUS

CN Benzamide, 4-(5-chloro-3-phenyl-2-pyridinyl)-N-[(5-methylpyrazinyl)methyl]-, monohydrochloride (9CI) (CA INDEX NAME)



● HCl

L7 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2003:737516 HCAPLUS

DOCUMENT NUMBER: 139:257284

TITLE: Cathepsin cysteine protease inhibitors and their therapeutic use

Updated Search

10554187

INVENTOR(S): Bayly, Christopher I.; Black, Cameron; Leger, Serge;  
Li, Chun Sing; McKay, Dan; Mellon, Christophe;  
Gauthier, Jacques Yves; Lau, Cheuk; Therien, Michel;  
Truong, Vouy-Linh; Green, Michael J.; Hirschbein,  
Bernard L.; Janc, James W.; Palmer, James T.;  
Baskaran, Chitra

PATENT ASSIGNEE(S): Merck Frosst Canada & Co., Can.; Axys Pharmaceuticals,  
Inc.

SOURCE: PCT Int. Appl., 282 pp.  
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003075836	A2	20030918	WO 2003-US6147	20030228
WO 2003075836	A3	20040715		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2477657	AA	20030918	CA 2003-2477657	20030228
AU 2003219953	A1	20030922	AU 2003-219953	20030228
US 2003232863	A1	20031218	US 2003-377377	20030228
EP 1482924	A2	20041208	EP 2003-716238	20030228
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
BR 2003008208	A	20050111	BR 2003-8208	20030228
CN 1638757	A	20050713	CN 2003-805181	20030228
JP 2005526753	T2	20050908	JP 2003-574112	20030228
US 2005240023	A1	20051027	US 2004-505796	20040825
NO 2004004207	A	20041124	NO 2004-4207	20041004
PRIORITY APPLN. INFO.:			US 2002-361818P	P 20020305
			US 2002-408704P	P 20020906
			WO 2003-US6147	W 20030228

OTHER SOURCE(S): MARPAT 139:257284

AB This invention relates to cysteine protease inhibitors  
R7(D)nCR6R7NR8CR3R4C(:O)NHCR1R2CN (R1-4 = H, (substituted)C1-6-alkyl or  
C2-6-alkenyl; R1 and R2 or R3 and R4 may be take together with the C atom  
to which they are attached to form a (substituted)C3-8-cycloalkyl or  
heterocyclic ring; R5 = H, (substituted)C1-6-alkyl; R6 =  
(substituted)aryl, heteroaryl, C1-6-haloalkyl, arylalkyl, heteroarylalkyl;  
D = (substituted)C1-3-alkyl, C2-3-alkenyl, C2-3-alkynyl, aryl, heteroaryl,  
C3-8-cycloalkyl, heterocyclyl; R7 = H, (substituted)C1-6-alkyl,  
C2-6-alkenyl, C2-6-alkynyl, C1-6-alkyloxy, etc.; R8 = H, C2-6-alkyl)  
including but not limited to, inhibitors of cathepsins K, L, S and B.  
These compds. are useful for treating diseases in which inhibition of bone  
resorption is indicated, such as osteoporosis.

IT 603140-97-2P

RL: SPN (Synthetic preparation); THU (Therapeutic use); BIOL  
(Biological study); PREP (Preparation); USES (Uses)

Updated Search

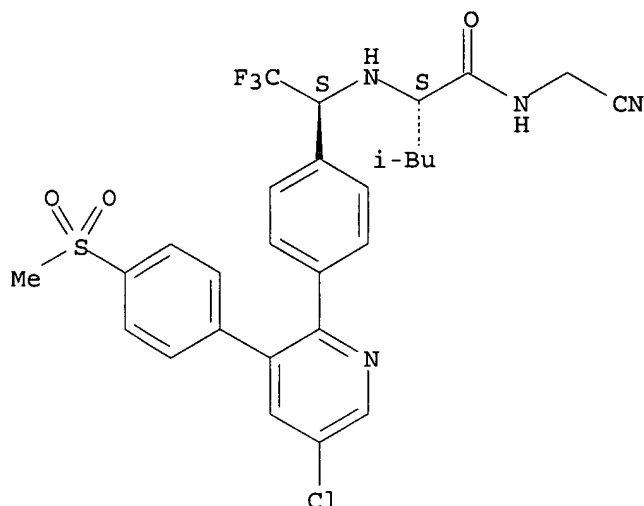
10554187

(cathepsin cysteine protease inhibitors and their therapeutic use)

RN 603140-97-2 HCAPLUS

CN Pentanamide, 2-[[[(1S)-1-[4-[5-chloro-3-[4-(methylsulfonyl)phenyl]-2-pyridinyl]phenyl]-2,2,2-trifluoroethyl]amino]-N-(cyanomethyl)-4-methyl-, (2S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



=> file caold

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
25.50	194.60

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE	TOTAL
ENTRY	SESSION
-3.00	-3.00

CA SUBSCRIBER PRICE

FILE 'CAOLD' ENTERED AT 14:42:25 ON 02 OCT 2006

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FILE COVERS 1907-1966

FILE LAST UPDATED: 01 May 1997 (19970501/UP)

This file contains CAS Registry Numbers for easy and accurate substance identification. Title keywords, authors, patent assignees, and patent information, e.g., patent numbers, are now searchable from 1907-1966. TIFF images of CA abstracts printed between 1907-1966 are available in the PAGE display formats.

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file supports REGISTRY for direct browsing and searching of all substance data from the REGISTRY file. Enter HELP FIRST for more information.

Updated Search

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=> d his

(FILE 'HOME' ENTERED AT 14:36:42 ON 02 OCT 2006)

FILE 'REGISTRY' ENTERED AT 14:39:04 ON 02 OCT 2006

L1 STRUCTURE UPLOADED

L2 1 S L1

L3 36 S L1 FULL

FILE 'HCAPLUS' ENTERED AT 14:41:10 ON 02 OCT 2006

L4 4 S L3

L5 4 S L3/THU

L6 2 S L5 AND DUGGAN, M?/AU

L7 2 S L5 NOT L6

L8 0 S L7 AND LINDSLEY, C?/AU

L9 0 S L7 AND WU, Z?/AU

L10 0 S L7 AND ZHAO, Z?/AU

L11 0 S L7 AND HARTNETT, J?/AU

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